Childhood lead exposure in the British Isles during the Industrial Revolution

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Abstract

Man-made toxin exposure is one of the defining characteristics of the second epidemiological transition. Our analysis of previous data shows that lead levels in tooth enamel above 0.87 ppm are characteristic of exposure to anthropogenic lead. In British prehistoric and Early Medieval populations very low lead concentrations have been observed, but Roman, later Medieval and Post-medieval populations show much higher levels, up to 90 ppm. Our measurements of lead concentrations within the tooth enamel of four 17th and 18th century populations from Coventry and London show no detectable association between lead exposure and cribra orbitalia (as a possible indicator of anaemia caused by plumbism), but do show population differences which we attribute to lower exposure of poor and rural people compared to rich and urban people. No differences in lead exposure by sex were found. Lead isotope ratios indicate that coal smoke was not a major contributor to lead exposure, but that ingested lead from artefacts is the most likely source. We show that the lead to which people were exposed in the post-medieval period has a similar average isotope ratio to that in the Roman period, but differs from early and later medieval periods.

Introduction

The second epidemiological transition, as originally characterised by Omran (1971) includes a shift "in mortality and disease patterns whereby pandemics of disease are gradually displaced by degenerative and man-made diseases as the chief form of morbidity and primary cause of death". Omran's characterisation has been widely accepted as describing a shift in causes of mortality from c.1840 to c.1920 in Britain and other industrialising nations (Barrett et al. 1998; McKeown 2009; Mooney 2007; Noymer and Jarosz 2008). Although historians have considered and debated the claim of a concomitant shift in morbidity and its relation to "man-made diseases" (Riley 2001), palaeopathologists working with human remains have paid less attention to it. In this paper we approach the question of one anthropogenic cause of morbidity; not by direct examination of the morbidity, but via measuring exposure to a toxin. We investigate the concentration of the toxin – lead – in the tooth enamel of eighteenth and nineteenth century individuals, using this as a proxy for lead exposure during childhood, and attempt to assess its impact on morbidity.

It has been shown using measurements on ice-cores that atmospheric lead pollution in the northern hemisphere has increased about 400 times between prehistory and the mid 1960s, with a major increase from about 500 BC, a decline in the second half of the first millennium AD and then a continuous increase during the second millennium AD (Hong et al. 1994). However, global atmospheric pollution is not the main route of exposure of humans, and is not directly reflected in human remains, which record local exposure to lead, although the peaks of exposure recorded in tooth enamel reflect the same trends in intensity of lead exploitation from the Neolithic to the early 16th century AD (Budd et al. 2004; Montgomery et al. 2010). Environmental and historical evidence indicates that there were further increases in lead usage in the post-medieval period. In the sixteenth century the increase seems to have been rapid, as Harrison (1587: 188) recorded that within living memory there had been "exchange of vessell, as of treene [i.e. wooden] platters into pewter".

Previous investigations of lead in human remains have not covered this post-medieval increase in exposure to lead.

Health effects of lead exposure

Lead poisoning (plumbism) causes a range of symptoms though the effects vary depending on the amount and duration of exposure, as well as the age of the individual exposed, with children having the greatest susceptibility to lead poisoning (Garrettson 1990; Putnam 1986). Acute poisoning is rare, and usually related to the ingestion of soluble lead salts; it causes death within one or two days if untreated. Chronic, long-term exposure to lead may cause gastrointestinal irritation ('dry belly-ache'), reduced libido and infertility, anorexia, anaemia, blue-lines on the gum-margins, peripheral neuropathy (wrist-drop), convulsions and encephalopathy (i.e. neural dysfunction) including impairment of visuospatial/visual motor functioning, short-term memory loss, and confusion and fatigue (Patrick 2006; Weisskopf et al. 2004). It has also been associated with gout due to impairment of the renal system (Matte et al. 1992). Plumbism causes anaemia by interfering with the action of various enzymes in the biosynthesis of haem, and through other effects which reduce production or increase destruction of red blood cells (Patrick 2006). It is thus primarily an iron deficiency anaemia but also has aspects of haemolytic anaemia. The level of lead in the blood is correlated directly with the severity of the anaemia (WHO 2010:24). The mechanisms by which infertility is caused differ in males and females. In males, libido and sperm count are reduced, probably via the disruptive effect of lead on the production of reproductive hormones including testosterone, and with no safe lower limit of exposure (Vigeh et al. 2010). In females, the main effect is through an increase in spontaneous abortion even at low exposures (Hertz-Picciotto 2000).

Subclinical poisoning, with symptoms not detectable in a clinical examination, is most common in childhood. Previously this has been associated with elevated levels of lead in the blood but recent work has suggested that the lower limit for effects is below the lowest level of 1 $\mu g dl^{-1}$ that has been studied, and there may be no safe lower limit (WHO 2010:25). Subclinical poisoning may result in developmental and behavioural changes, including reduced mental acuity (Dietrich et al. 2001; Lanphear et al. 2000; Needleman 2004) and a tendency towards behavioural abnormality (Dietrich et al. 2001).

Childhood exposure starts with exposure in the womb, as foetal blood lead levels are very close to maternal levels. Exposure to maternal lead will reduce with breast-feeding as little lead is transmitted in breast-milk, but this is offset by a higher rate of absorption in the immature gut and the hand-to-mouth behaviour of young children can lead to oral exposure to lead-bearing objects (WHO 2010:22). In the nineteenth century, high infant mortality, and poor growth and health which would now be termed 'failure to thrive', were believed to be caused by poor nutrition (e.g., Davis 1817). However, the symptoms of failure to thrive are very similar to the symptoms of low level lead poisoning, from the low birth weight to the continuing lack of physical and neurological development. It is possible therefore that one, unrecognised, cause of the widespread failure to thrive of infants in the nineteenth century was lead poisoning, whether maternally transmitted or post-natally acquired.

Lead pollution in the eighteenth and nineteenth centuries

The industrialisation that occurred in England during the eighteenth and nineteenth centuries has been characterised as the transition from an 'organic' to 'mineral' economy, with concomitant increase in coal use (Daunton 1995; Wrigley 1988). It is the smoke and fog resulting from coal use that dominates popular images of the period (thanks to the descriptions of contemporary authors such as Dickens), but other sources of pollution, including lead pollution, were also increasing. The transition from an 'organic' to 'mineral' economy involved not only fuel, but also the replacement of wooden vessels with inorganic ones, including lead-glazed ceramics and lead and pewter vessels. Acidic materials stored in lead containers would rapidly leach lead into the food or beverage (Richards 1999). Although by the 1750s there was some understanding of the dangers associated with acidic materials in lead-glazed or bearing vessels (e.g., Hardy 1778; Lind 1754), this only slowly reduced the frequency of lead poisoning due to the exposure of alcoholic drinks to lead or lead glazed vessels (Handler et al. 1986). Deliberate contamination of food with lead, either as a sweetener or colorant, was a well-known health issue in the mid nineteenth century (Drummond and Wilbraham 1939:292). Likewise, the utilisation of lead to sweeten wine continued into the nineteenth century (Eisinger 1982).

The burning of coal was a possible source of lead exposure as well. Farmer et al. (1999) estimated that in 1830 between 9% and 33% of atmospheric lead released in the UK was from coal, with the rest from lead smelting, increasing to between 11% and 38% by 1855. Away from lead production areas therefore, the atmospheric releases from coal are likely to have been the majority of atmospheric lead releases. Today, however, exposure by inhalation is reckoned to be a minor source of lead poisoning because the particles produced by domestic use of coal are usually too large to be inhaled (WHO 2010:18). Whether coal burning could have been a contributing factor to past lead exposure is therefore unclear.

Occupational lead exposure was also acknowledged for deleterious health effects, if not specifically plumbism, from the seventeenth century onwards (Weeden 1984). By the nineteenth century those professions that were most susceptible to lead poisoning were listed by Thackrah (1831), including miners, ironworkers, founders, potters, brass workers and solderers. However, this paper's examination of tooth enamel confines the lead exposure that can be investigated to childhood.

Lead exposure was therefore widespread in all ranks of society, but due to the cost of items such as pewter, lead crystal and wine, the rich were more likely to experience long-term lead exposure than the poor (except those with specific occupational exposure), and lead was more widely used in urban than rural contexts. Indeed, those living on a subsistence diet in rural areas may have escaped exposure to the major sources of anthropogenic lead. This is in contrast to the present day, where lead poisoning is predominantly a disease of the poor (WHO 2010:35).

Study sites

All the study sites were selected within projects designed primarily to investigate migration rather than lead exposure per se. The sample is therefore not optimised to relate lead exposure to social factors. Their locations are shown in Figure 1.

[**FIGURE 1: Locations of the sites and of British lead-ore fields.** Outline reproduced from Ordnance Survey map data by permission of the Ordnance Survey © Crown copyright 2010. The approximate locations of the London sites are shown overlain on Rocque's map of 1741-45, depicting London at the very start of the period considered. Major lead ore fields' locations are shown approximately, based on Rohl (1996).]

Coventry

In 1801 Coventry was a regional city of some 16,000 inhabitants. The population had grown rapidly over the preceding century from about 6700 in 1694 and more than doubled again by 1851 to over 36,000, with migration a major factor in this expansion (Lancaster and Tomlinson 1969:5). The primary industries of the city were ribbon-weaving (though this declined and virtually disappeared between 1840 and 1860) and watch-making (Lancaster 1969:168-172).

The cemetery investigated was the churchyard of the parish of Holy Trinity, Coventry, with remains recovered from an area representing an extension in 1776 of that cemetery over the demolished nave and aisles of the former St Mary's cathedral. The cemetery was formally closed in 1856, though burials had become increasingly rare in the preceding years, with only 38 since 1849. Some additional burials within family tombs continued until as late as 1890. Excavations ahead of redevelopment in 1999 entailed the removal of 1,706 articulated human skeletons (Rylatt and Mason 2003). The majority of remains were reburied, but some were retained for further study. The ten individuals sampled represent retained skeletons which had associated coffin plates giving partial or complete identification; their dates of death range from 1825 to 1847 and their dates of birth c.1780 to c.1823. Although Coventry was not a very wealthy city, the selection of skeletons with coffin plates will have biased the sample towards higher social classes.

Chelsea

In the early eighteenth century, Chelsea was a village on the river Thames and the main road west from the City of London, about 3 miles from Westminster, and not within the urban area. In 1674 it is recorded as having only 172 houses, but by 1795 this had increased to 1350 households. With a population of less than 12,000 in 1801 it was smaller than Coventry, but by 1851 this had increased to almost 54,000 and it was included within the expanding metropolitan area of London (Croot 2004; Rudé 1971:9-10). Chelsea had a reputation as the residence of the upper-classes – Defoe (1724) described it as a "town of palaces" – but the eighteenth century saw development of a service and pleasure industry due to those upper-class residents and Chelsea's favourable location close to the Cities of Westminster and London (Cathcart-Borer 1973; Insley and Croot 2004:166; Rudé 1971:9-10). The epitome of this was the Chelsea Bun House run by the Hand family. The economy of Chelsea was diverse, with significant areas of market garden persisting into the nineteenth century and supplying the needs of London (Bryan 1869). In the eighteenth century there were porcelain manufactories and until c.1825 foundries for bell- and weapon-manufacture (Croot and Insley 2004:158-160).

Excavations in 2000 investigated part of the graveyard of Chelsea Old Church. A total of 285 skeletons were recovered, mostly from the eighteenth and nineteenth

centuries. The majority were interred in wooden coffins though some were in lead coffins, including five of those considered here (Cowie et al. 2008). Twenty-four individuals were sampled with a preference for those identifiable from coffin plates, so that nine identified individuals are considered here.

Lukin Street, Whitechapel

Since the seventeenth century, Whitechapel and London's docklands have been home to successive waves of immigrants seeking unskilled work and cheap accommodation (White 2007:134,152). With a long-established Irish community, the population here was swelled in the 1840s first by slum clearances in St Giles ("Little Ireland") to build New Oxford Street, and then by the mass migration of the very poor escaping the Great Irish Famine of 1847-51 (White 2007:32). The population density in Whitechapel was fifteen times that of Chelsea in 1841 and overcrowding and poor sanitation coupled with extreme poverty led to this area having a lower life expectancy than other areas of London (Graham 1843).

Excavations at the cemetery of the Catholic Mission of St Mary and St Michael, Whitechapel (LUK04) provided a sample of 705 individuals (268 adults and 437 subadults) buried between 1843 and 1854. Epigraphic evidence suggests this burial ground served a population chiefly of Irish descent, some of whom came to England during the Great Famine of 1847-8 from the poorest rural areas of Ireland (Powers 2008). This is corroborated by documentary evidence that the Catholic Mission served a first generation Irish community (Powers 2008). In order to identify first generation migrants, 120 individuals have been studied using a suite of stable isotope ratios from hair, bone and teeth, of whom forty-five were sampled for enamel lead concentrations.

Golden Lane, London

Golden Lane, on the northern side of the City of London, was described by a police sergeant as a "bad, ruffianly, thievish place" (Mayhew 1861:237). A poor area with a large Irish community, it was noted for the number of taverns. Even as late as 1874, it was described as 'the "slummiest" of slums' (Greenwood 1874:19). The surrounding area, however, was the focus for non-conformists such as the Quakers, and John Wesley's Chapel was established nearby in 1778 (Connell and Miles 2010).

The City Bunhill Burial Ground was on the site of a former brewery. This dissenters' cemetery was in use from October 1833 to August 1853, and in just over 20 years more than18,000 burials appear in the registers. A sample of addresses suggests that more than half were not from the local area (i.e. within 500m) and that people were brought to the cemetery for burial from a wide area of London (Connell and Miles 2010). During excavations in 2006, the remains of 239 individuals were excavated by Museum of London Archaeology: osteological analysis revealed high infant mortality and disease patterns consistent with other low socio-economic groups in London. The thirteen legible coffin plates from the excavation do not contain any Irish surnames, although as a non-parochial cemetery it is likely that Catholics will have been buried here (Connell and Miles 2010).

Materials and Methods

The data used in this study were collected within two separate PhD projects at different universities, using slightly different protocols, but the methods yield commensurate results.

Of the skeletal materials available, enamel was selected as the tissue for analysis due to its resistance to diagenesis and its retention of biogenic concentrations and isotope ratios for lead (and for parallel studies of strontium), in contrast to bone and dentine which often exhibit changes due to the burial environment (Budd et al. 2000; Millard 2006; Trickett et al. 2003). For the majority of skeletons we selected second premolar or second molar teeth, though the lack of preserved teeth meant that other teeth were selected for a minority of individuals. The enamel of the second premolar and the second molar is formed between approximately 2.5 and 6.5 years of age (Moorrees et al. 1963), so, in the main, our results relate to exposure to lead in this period of childhood.

Lukin Street and Golden Lane: For each tooth, the outer surface of the enamel was removed to a depth of >100μm with a tungsten carbide burr. Enamel samples were cut from the tooth with a flexible dental saw, and all saw-cut surfaces, and any adhering dentine then rigorously cleaned with a tungsten carbide bur. After cleaning with dilute acid, 5-10 mg of enamel was rinsed with ultrapure water, dried, and weighed. The enamel was then dissolved in acid and lead concentrations were measured by inductively coupled plasma mass spectrometry (ICP-MS) on an Agilent 7500cx quadrupole mass spectrometer MS at the British Geological Survey, Nottingham. Full details of the protocols are given in Montgomery et al (2010). The reproducibility of the lead concentration data was ± 10% (2σ).

Chelsea and Coventry: Each tooth was half-sectioned and the dentine removed with a dental burr. The outermost 200-300μm of the internal, external, and cut surfaces of the enamel cap were removed to eliminate any surface contamination. After cleaning in ultrapure water and acetone, approximately 50 to 100mg of enamel was dissolved acid and spiked with ^{208}Pb. Lead was purified using ion exchange chromatography. Concentrations were determined by isotope dilution using Thermal Ionisation Mass spectrometry on a Finnigan MAT262 multi-collector mass spectrometer and the lead isotope compositions were determined using a VG Elemental Axiom MC-ICPMS. The data were normalised to the reported values of NBS 981 (Thirlwall 2002). Full details of these protocols are given by Trickett (2006). Lead concentrations were measured with a precision better than ±10%.

As neither concentrations nor isotope ratios of lead are expected to be normally distributed, non-parametric statistical tests were used. In all comparisons Levene's test based on medians was used to compare variances of the groups and if no significant difference was found, a Mann-Whitney test (for two groups) or Kruskal-Wallis test (for more than two groups) was used to compare central tendency. Where significant differences were found in comparing multiple groups, then pairwise Mann-Whitney tests (or in the case of unequal variances, Kolmogorov-Smirnov tests) were used to establish which groups differed. All calculations were performed using the Palaeontological Statistics Package (PAST) (Hammer et al. 2001).

Results

The results of this study are shown in Tables 1 and 2.

[**TABLE 1: Sample details and lead concentrations.** Blank cells indicate a lack of data. Notes: 1. Site codes: COV: Coventry St Mary; GDA: Golden Lane; LUK04: Lukin Street; OCU00: Chelsea Old Church. 2. Using the FDI system. 3. Age at death from coffin plate is indicated by *, other ages are osteological estimates.]

[**Table 2: Lead isotope ratios for Chelsea and Coventry individuals.** %RSD is percent relative standard deviation.]

Identifying 'natural' levels of exposure

Before evaluating the extent of anthropogenic exposure to lead, it is necessary to establish what range of lead concentrations in tooth enamel can occur naturally. Figure 2 shows the dataset compiled by Montgomery et al. (2010) together with our data from Chelsea and Coventry, plotted as concentration versus $^{207}Pb/^{206}Pb$. The spread of lead isotope ratios clearly decreases with increasing lead concentration. This phenomenon is attributed to high levels of exposure occurring with lead-rich anthropogenic sources with a limited isotopic range, and low levels of exposure deriving from isotopically-diverse natural sources. This inverse correlation of isotopic ratio with concentration has therefore been termed "cultural focussing" (Montgomery et al. 2005).On the basis of a visual inspection of a similar figure, Montgomery et al (2010:212) suggested that lead concentrations in excess of about 0.5 ppm should be regarded as having an anthropogenic component as this is the point at which cultural focussing becomes apparent. Here we extend the analysis with a more formal investigation to define a suitable cut-off point above which we regard the lead content of human tooth enamel to have an undoubted technological contribution. If the data of Figure 2 are partitioned into two groups on the basis of their lead concentration, and the variance of the higher concentration group is plotted as a function of the cut-off between the two groups (Figure 3), it is clear that there are major changes in variance at 0.87 ppm and 30 ppm. We therefore take values at and above 0.87 ppm as falling in a 'cultural' category where the anthropogenic component of lead exposure dominates. Conversely, values at and below 0.68 ppm are considered primarily natural, though some anthropogenic component cannot be ruled out. We have no isotopic data for concentrations between 0.68 and 0.87 ppm to better define the upper limit of natural exposure. All Neolithic and Bronze Age humans from the dataset compiled by Montgomery et al. (2010) fall within the 'natural' category. The change in variance at 30 ppm is discussed below.

[**Figure 2: Lead concentration compared with $^{207}Pb/^{206}Pb$ ratio.** Note the logarithmic scale for concentration. The solid horizontal line is our lower limit of 0.87 ppm for undoubted 'cultural' lead exposure.]

[**Figure 3: Variance of $^{207}Pb/^{206}Pb$ for samples above a given Pb concentration.** Note that both scales are logarithmic. Labels show concentrations at points between which changes in variance occur.]

Statistical comparisons

Within our dataset, Lukin Street is the only site where people display 'natural' levels of lead, with two individuals below 0.68 ppm, and one ambiguous at 0.77 ppm. A Kruskal-Wallis test comparing the four sites shows that there is a very significant difference between sites in lead concentrations (H=19.34, p=0.0002), and pairwise Mann-Whitney tests indicate that the difference is between Lukin Street and the other sites; Chelsea, Coventry, and Golden Lane do not differ significantly from one another.

Comparison of lead concentrations by sex reveals no statistically significant difference (Kruskal Wallis test, H=14.24, p=0.23). Subdividing by sex and site reveals a significant difference (Kruskal Wallis test, H=19.2, p=0.0018), which pairwise Mann-Whitney tests indicate arises because males and females from Lukin Street differ from males and females from Chelsea.

The effect of social status on lead exposure may be investigated by comparisons between sites or between individuals. At the individual level the only indicator of social status that we have is the presence or absence of coffin plates. For the whole dataset there is no difference between those with and without coffin plates (Mann Whitney U=816, p=0.70).

Iron-deficiency anaemia has been identified as a major cause of cribra orbitalia, a pathology which usually occurs in childhood and is observed in skeletal material as a pitting of the bone of the orbits, which still remains visible in adults (Stuart-Macadam 1989). Individuals from all the sites have been scored in a consistent manner for the extent of cribra orbitalia using the system of Stuart-Macadam (1991). If lead exposure was sufficient to cause significant iron-deficiency anaemia then we would expect a correlation of the extent of cribra orbitalia with lead concentration. Comparison of lead concentrations between individuals showing cribra orbitalia and those without cribra orbitalia showed no significant difference (Mann Whitney U=536, p=0.67), nor was there any correlation between the severity score for cribra orbitalia and lead concentration (Spearman's rho=0.01, p=0.91).

Comparison of our isotope data for Chelsea and Coventry with the dataset compiled by Montgomery et al. (2010) shows that the $^{207}Pb/^{206}Pb$ ratio for humans with 'cultural' lead concentrations differs in variance between periods (Levene's test, p=0.0018, see also Fig. 2) and comparisons using pairwise Kolmogorov-Smirnov tests show that post-medieval individuals' ratios differ significantly from those of early and late medieval people, but there is no significant difference when comparing post-medieval and Roman individuals.

Discussion

Lead exposure

Reported lead concentrations in human tooth enamel from Britain, from this and previous studies, vary by a factor of approximately 30000, from 3 ppb to over 90 ppm (Figure 2). Variability resulting from natural exposure may be up to 300 times, but at least a further hundred-fold variation in concentration arises from cultural exposure to lead. Within that cultural variation, the post-medieval populations presented in this

paper show a three-fold increase in maximum lead concentrations compared to the maxima reported by Montgomery et al. (2010) for Roman (30.1ppm) and Viking (31.6 ppm) populations. With 24% (20/83) of our post-medieval individuals exceeding the maximum for previous periods, and only 4% (3/83) having 'natural' levels of exposure, the consistent and high exposure of post-medieval populations is clear. The individuals above 30 ppm come from all four of our sites, but a higher proportion of individuals from Chelsea (10/23) and Golden Lane (3/5) occur in this group than from Coventry (1/10), and Lukin Street (7/45) (χ^2-test, p=0.01), concordant with the expectation that higher status populations had greater lead exposure.

Gulson (1996) reported enamel lead concentrations up to 30 ppm in children who had ingested dust from lead paint, but these were extreme and even those who had high exposure from living in a lead-mining community typically had values of 2-10 ppm. The values exhibited by our post-medieval populations are therefore high to extremely high compared to modern populations in which the effects of lead are well-documented.

Relating this high lead exposure to morbidity is much more difficult than simply identifying it. Sampling tooth enamel means that the measurement relates to the average lead exposure during the period of formation of the enamel during childhood, which is at least two or three years. Any acute exposure to lead will therefore be averaged out in our samples, and the total variability in exposure to lead must be much greater than we observe. The literature on clinical manifestations of lead poisoning is based around levels of lead in blood, but there are very few studies relating blood lead levels to enamel levels, and the findings of such studies are difficult to interpret, as the samples taken must necessarily relate to different periods of life.

Direct inference of morbidity from enamel lead concentrations is therefore not possible and we must therefore seek other lines of evidence. Corroboration of many of the effects of lead poisoning using the skeletal or historical records is not easy, but two of them may be detectable: infertility/reduced libido and anaemia. Of the named adults in our dataset we currently only have information on the reproductive success of eight (Table 1). While within that eight the three with the lowest concentrations did have children, the dataset is really too small to establish any effect and the difference in lead concentrations for those with and without children is not significant (Mann Whitney U=3, p=0.23). More historical data is needed to investigate this and it would be preferable to be able to consider separately male and female reproductive success. In addition it would be necessary to compare the historical data on individuals to more general data on the period which suggests that the percentage of adults who never married was 9-12% in the eighteenth to early nineteenth centuries (Schofield 1985) and primary sterility occurred in 7% of marriages (Wrigley et al. 1997:384).

Although lead exposure is known to cause iron-deficiency anaemia, and cribra orbitalia has been associated with anaemia (Stuart-Macadam 1989), we have not observed any association between childhood lead concentrations as recorded in enamel and the occurrence or severity of cribra oribitalia. This may be because of small sample sizes, but is also consistent with recent work which has proposed other aetiologies for cribra orbitalia (Walker et al. 2009).

As anticipated from the historical record, there appear to be differences by social class, with the clearest comparison being between Lukin Street in the deprived area of Whitechapel and well-to-do Chelsea. However this is not a simple comparison as the Chelsea sample is slightly earlier and the Lukin Street sample probably includes a high proportion of immigrants from rural Ireland, so this is more likely to be due to rural-urban differences. This suggestion is supported by the small sample from Golden Lane who were probably poor but not Irish immigrants and who exhibit higher lead levels than the Lukin Street population, and by the fact that the intermediate social status group from Coventry also have higher lead levels than the Lukin Street sample. The economy of rural Ireland was at a subsistence level, and would have involved less use of lead, and the burning of peat rather than coal, so that the exposure of the population to lead was as low as in prehistoric periods. The individuals from Lukin Street exhibiting high lead levels seem less likely to be first generation immigrants from rural areas.

Sources of lead exposure

Figure 4 compares the isotope ratios of lead from the individuals from Coventry and Chelsea with the ratios reported in the literature for major lead ore sources and coal from England and Wales. In comparison to the ratios from coal, the humans plot in an unlikely position for an average of coal sources and at and beyond the upper end of the distribution of values from the Durham coalfield which was the major source of coal for London at this time (Daunton 1995:220). It seems unlikely therefore that coal smoke played a major part in the exposure of children to lead in these populations.

The group with high lead concentrations also have a narrower range of lead isotope ratios than in previous periods, which might indicate either a reduction in the range of ore sources exploited or further cultural focussing of lead isotope ratios by recycling of lead. As the eighteenth and early nineteenth centuries were a time of expansion in lead production and in the number of mines, a reduction in the range of sources seems unlikely. When compared to the lead isotope ratios of lead ores from the three major ore-fields, most of the tooth enamel values cluster over the southern Pennines values and between the Bristol/Mendips and north Pennine values. During the period for which there are good production figures, the North Pennine ore-field dominated production, with 35-40% of all UK production in the period 1851-1881 (computed from Burt 1984:194). Although this is slightly later than the period of exposure of the individuals studied here, the relative productivity is unlikely to have changed much. It therefore seems unlikely that the south Pennine ore-field was the major source of lead to which people were exposed, even though this ore-field is not as well characterised as the other two. Instead, the tight grouping of values implies that humans were being exposed to lead in such a way that the isotope ratio is an average of English lead ores. This could have been via exposure to a variety of items containing lead derived from different ores, or the use of recycled lead which would average the isotope ratios of lead in circulation and yield a consistent isotope ratio for all lead exposure.

Three individuals stand out as having distinctly higher lead isotope ratios, and two of these are very close together and fall amongst a group of lead-ore samples which derive from the Mendips. One of these individuals is Thomas Langfield (CHE147) who is known to have spent his childhood in Somerset, where one might expect the primary source of anthropogenic lead to be the Mendip mines. The averaging seen

in the majority of the sample investigated might therefore not be characteristic of the country as a whole, but reflect the nature of urban exposure to lead.

[**Figure 4: $^{208}Pb/^{206}Pb$ v $^{207}Pb/^{206}Pb$ for post-medieval human tooth enamel compared to the major lead ore fields and coal from England and Wales.** Data sources for mineral isotope compositions: Bristol/Mendips lead (Rohl 1996), North Pennines lead (Rohl 1996; Scaife et al. 2001; Shepherd et al. 2009), Southern Pennines lead (Rohl 1996), Durham coal (Farmer et al. 1999; Shepherd et al. 2009), other England and Wales coal (Farmer et al. 1999).]

The immediate source of exposure cannot easily be identified without isotopic analysis of contemporary lead, pewter, and lead-glaze. This would allow us to identify whether there are multiple isotopically distinct lead sources averaged in the human body, or whether the recycling of lead has averaged the ore-source compositions before exposure. Nevertheless the fact that early and late medieval people differ in isotopic composition from Roman and post-medieval people indicates that the balance of ore-sources being exploited differs between periods. The post-medieval period saw a return to the exploitation of sources, or to a balance of sources, which was similar to that of the Roman period, when lead use and exposure reached its pre-industrial peak.

Conclusion

Our results show clearly that the post-medieval populations examined here include individuals who exhibit higher levels of childhood lead exposure than any others reported in the literature. Although it is difficult to correlate this exposure with morbidity, we do not doubt that there must have been effects from this lead exposure, with the most likely effects being anaemia, reduced mental acuity and, if the body burden of lead continued into adulthood, reduced fertility. The source of the exposure was primarily lead derived from lead ores rather than lead released by the burning of coal, despite the contribution that the latter made to atmospheric releases of lead. Rural and poor populations were less exposed than rich and urban ones, but there are no differences by sex, which suggests that boys and girls were exposed to similar degrees.

In the context of the second epidemiological transition, we can clearly corroborate the claim that exposure to environmental pollution (in this case lead) increased with industrialisation and that exposure reached levels unheard of before or since that time. From skeletal material and historical records it is difficult to establish what specific morbidities arose from that exposure, though comparisons with studies in the late twentieth century suggest that they must have occurred. However, contrary to those studies, we find that wealth and urban dwelling were major drivers of exposure during the eighteenth and early nineteenth centuries, which supports the assertion that the change in morbidity of second epidemiological transition was driven by industrialisation.

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Table 1: Sample details and lead concentrations

Blank cells indicate a lack of data.

1. Site codes: COV: Coventry St Mary; GDA: Golden Lane; LUK04: Lukin Street; OCU00: Chelsea Old Church.

2. Using the FDI system.

3. Age at age at death from coffin plate is indicated by *, other ages are osteological estimates.

Site code[1]	Skeleton	Tooth[2]	Sex	Cribra orbitalia	Year of Death	Age at Death[3]	Title	Forename	Surname	Coffin plate	Offspring?	Pb ppm
COV	50	14	F	N		43*		…	…ein…	Y		29.92
COV	76	25	F	N	1827	30*		Eliza(beth)	Burton	Y		9.51
COV	417	44	F	Y	1847	60*		Sarah	Green	Y		7.71
COV	433/4	35	F		1825	22*		Hannah	Denney	Y		18.40
COV	516	37	M	N	1845	65*		…	Cooper	Y		
COV	672	47	F		1844	30*		Eliza	Sparkes	Y		32.04
COV	808	44	M		1846	48*		William	Wagstaffe	Y		26.86
COV	866	47	F	N	1846	25*		Harriet	Parsons	Y		21.38
COV	978	44	M		1846	61*		James	Brown	Y		16.96
COV	1248	37	M	Y	1842	19*		John	Chattaway	Y		9.18
GDA06	522											31.14
GDA06	744											7.46
GDA06	757(H)											26.55
GDA06	837(H)											66.10
GDA06	991(H)											32.27
LUK04	13	37	F	N		early middle adult						7.88
LUK04	47	17	U	Y		18 to 25						92.22
LUK04	288	45	M	N		early middle adult						0.87
LUK04	413	16	M	Y	1850	12 to 18		John	Crawley	Y		26.85

LUK04	419	46	F	Y		12 to 18			y	Y	16.21
LUK04	557	35	F	N		late middle adult			….ona…oe	Y	6.27
LUK04	597	17	F	N	1852	late middle adult		Bridget	McNally	Y	21.93
LUK04	633	27	M	N		late middle adult					1.79
LUK04	755	27	M	N		late middle adult					6.37
LUK04	813	27	F	N		early middle adult					59.96
LUK04	833	27	M	N	1847	late middle adult		John	Berry	Y	2.01
LUK04	840	17	F	N		old adult					2.46
LUK04	848	17	M	Y		early middle adult		Timothy	Sullivan	Y	2.12
LUK04	873	17	F	N		late middle adult	Mrs	…	….acklin	Y	0.47
LUK04	881	27	M	N		late middle adult					0.77
LUK04	903	45	M	Y	1851	late middle adult		Michael	Ryan	Y	2.84
LUK04	913	35	M	Y		late middle adult					3.38
LUK04	948	35	M	N		early middle adult		Keon		Y	5.00
LUK04	1012	27	M	N		late middle adult					37.07
LUK04	1014	17	M	Y	1845	early middle adult		Alexander Henry	Creamer	Y	26.35
LUK04	1031	17	F	N	1847	late middle adult		Bridgett	Muldary	Y	2.77
LUK04	1041	17	F	N		early middle adult					2.72
LUK04	1081	37	M	N		old adult					10.45
LUK04	1113	27	M	Y		late middle adult					0.63
LUK04	1129	17	F	N	1851	early middle adult				Y	54.13
LUK04	1142	27	F	N		early middle adult	Mrs	Jane	Su…..	Y	2.34
LUK04	1162	27	F	Y	1844	late middle adult				Y	1.84
LUK04	1174	17	F	N		18 to 25	Miss			Y	0.91
LUK04	1210	27	M	Y		late middle adult					1.27
LUK04	1220	45	F	N		old adult					6.84
LUK04	1222	27	M	N		early middle adult		P	Sullivan	Y	23.11
LUK04	1282	33	F	N	1847	old adult				Y	7.32
LUK04	1290	17	M	N	184-	late middle adult				Y	71.17

LUK04	1312	43	F	N	1845	late middle adult		Georgiana	Neale	Y		30.00
LUK04	1314	27	M	Y		old adult						1.40
LUK04	1337	17	M	N	1848	old adult		John	Regan	Y		4.77
LUK04	1348	35	M	N	1846	early middle adult		Miguel	Penethera	Y		9.72
LUK04	1363	17	M	N		old adult						1.63
LUK04	1396	17	M	Y		late middle adult	Mr	Jam..		Y		39.29
LUK04	1404	17	F	N		early middle adult		Jul…	…oll	Y		18.33
LUK04	1430	27	M	Y	1845	late middle adult	Mr	C	Hart	Y		2.02
LUK04	1459	27	M	N	1846	18 to 25				Y		18.06
LUK04	1476	35	F	N		late middle adult		Bridgett	Hi	Y		2.51
LUK04	1495	27	F	Y		12 to 18	Miss	So…	Fl….ery	Y		33.02
LUK04	1567	17	F	N		late middle adult						4.14
OCU00	18	35	F	Y		36-45						82.42
OCU00	19	35	F	N		>45						9.42
OCU00	31	16	F	N		36-45						4.65
OCU00	35	45	M	N	1821	60*	Mr	Gideon Richard	Hand	Y	N	75.04
OCU00	39	35	F			36-45						44.75
OCU00	104	37	F	N		36-45						50.84
OCU00	147	44	M	N	1808	67*	Mr	Thomas	Langfield	Y	N	14.03
OCU00	161	35	F	Y		18-25						70.25
OCU00	285	37	M	N		36-45						12.49
OCU00	392	35	F	Y		18-25						42.69
OCU00	552	17	F	N		>45						19.39
OCU00	654	17	M	N	1827	78*	Mr	Thomas	Long	Y	Y	40.25
OCU00	697	15	F	N		>45						5.01
OCU00	713	18	M	N	1822	68*	Esq	John	Long	Y	N	83.44
OCU00	744	15/25	M	N	1793	70*	Mr	John	Long	Y	Y	12.96
OCU00	750	15/25	F	N		>45						28.95
OCU00	792	25	F	N	1807	68*	Mrs	Milborough	Maxwell	Y	Y	83.42

OCU00	841	37	F	Y		>45					27.88
OCU00	856	25	M	N		26-35					37.06
OCU00	918	37	F	N		>45					27.46
OCU00	980	35	F	N	1806	54*	Sarah	Adams	Y	Y	6.04
OCU00	990	37	F	N	1781	32*	Charity	Adams	Y	Y	13.29
OCU00	994	35	M	N		36-45					19.80
OCU00	1051	36/46	U		179?	1-5		Collon/ Collum/ Collins?	Y		23.60

6

7

Table 2: Lead isotope ratios for Chelsea and Coventry individuals.

%RSD is percent relative standard deviation

Site code	Skeleton	$^{206}Pb/^{204}Pb$	%RSD	$^{207}Pb/^{204}Pb$	%RSD	$^{208}Pb/^{204}Pb$	%RSD	$^{207}Pb/^{206}Pb$	%RSD	$^{208}Pb/^{206}Pb$	%RSD
COV	50	18.4495	0.003	15.6286	0.005	38.4143	0.007	0.84710	0.002	2.08215	0.005
COV	76	18.4218	0.004	15.6309	0.006	38.3971	0.008	0.84849	0.002	2.08447	0.005
COV	417	18.4014	0.003	15.6083	0.003	38.3301	0.004	0.84820	0.001	2.08300	0.002
COV	433/4	18.4373	0.002	15.6239	0.003	38.4015	0.003	0.84739	0.001	2.08279	0.001
COV	516	18.4456	0.004	15.6254	0.006	38.4142	0.007	0.84714	0.002	2.08267	0.005
COV	672	18.4314	0.003	15.6193	0.003	38.3846	0.004	0.84744	0.001	2.08253	0.002
COV	808	18.4360	0.002	15.6253	0.002	38.3944	0.003	0.84756	0.001	2.08264	0.001
COV	866	18.4415	0.007	15.6298	0.008	38.4233	0.009	0.84752	0.002	2.08352	0.003
COV	978	18.4438	0.002	15.6222	0.002	38.3967	0.003	0.84702	0.001	2.08182	0.001
COV	1248	18.4183	0.002	15.6131	0.003	38.3719	0.004	0.84771	0.001	2.08336	0.002
OCU00	18	18.4232	0.006	15.6197	0.006	38.3972	0.007	0.84782	0.002	2.08411	0.003
OCU00	19	18.4286	0.008	15.6142	0.009	38.3759	0.012	0.84727	0.002	2.08232	0.005
OCU00	31	18.3766	0.009	15.6168	0.011	38.3471	0.014	0.84987	0.002	2.08686	0.005
OCU00	35	18.4460	0.007	15.6179	0.009	38.3936	0.012	0.84667	0.001	2.08138	0.004
OCU00	39	18.4206	0.008	15.6118	0.009	38.3731	0.012	0.84751	0.001	2.08307	0.005
OCU00	104	18.4398	0.005	15.6153	0.006	38.3829	0.009	0.84683	0.001	2.08145	0.004
OCU00	147	18.3745	0.005	15.6163	0.006	38.3465	0.008	0.84991	0.002	2.08694	0.003
OCU00	161	18.4188	0.005	15.6173	0.006	38.4069	0.008	0.84790	0.001	2.08517	0.003
OCU00	285	18.4396	0.005	15.6168	0.006	38.3743	0.009	0.84691	0.001	2.08105	0.003
OCU00	392	18.4369	0.005	15.6264	0.006	38.4388	0.009	0.84758	0.002	2.08496	0.005
OCU00	552	18.4445	0.005	15.6271	0.006	38.4101	0.010	0.84730	0.002	2.08262	0.005
OCU00	654	18.4520	0.005	15.6277	0.006	38.4290	0.009	0.84692	0.002	2.08267	0.005
OCU00	697	18.4357	0.007	15.6286	0.008	38.4212	0.010	0.84774	0.002	2.08420	0.006
OCU00	713	18.4523	0.004	15.6302	0.004	38.4388	0.006	0.84707	0.002	2.08316	0.004

OCU00	744	18.4441	0.006	15.6291	0.006	38.4221	0.006	0.84739	0.002	2.08314	0.002
OCU00	750	18.4402	0.006	15.6285	0.006	38.4174	0.006	0.84752	0.002	2.08332	0.002
OCU00	792	18.4332	0.006	15.6324	0.006	38.4403	0.006	0.84806	0.002	2.08536	0.002
OCU00	841	18.4502	0.006	15.6274	0.006	38.4238	0.006	0.84700	0.002	2.08261	0.002
OCU00	856	18.4524	0.003	15.6286	0.004	38.4157	0.005	0.84696	0.002	2.08192	0.003
OCU00	918	18.4541	0.004	15.6289	0.004	38.4173	0.005	0.84695	0.002	2.08192	0.003
OCU00	980	18.4058	0.003	15.6293	0.004	38.3932	0.006	0.84914	0.002	2.08594	0.004
OCU00	990	18.4069	0.004	15.6172	0.003	38.3735	0.006	0.84844	0.002	2.08475	0.004
OCU00	994	18.4495	0.007	15.6260	0.008	38.4163	0.010	0.84699	0.002	2.08230	0.004
OCU00	1051	18.4364	0.004	15.6312	0.005	38.4203	0.007	0.84785	0.002	2.08397	0.005

Figure 1

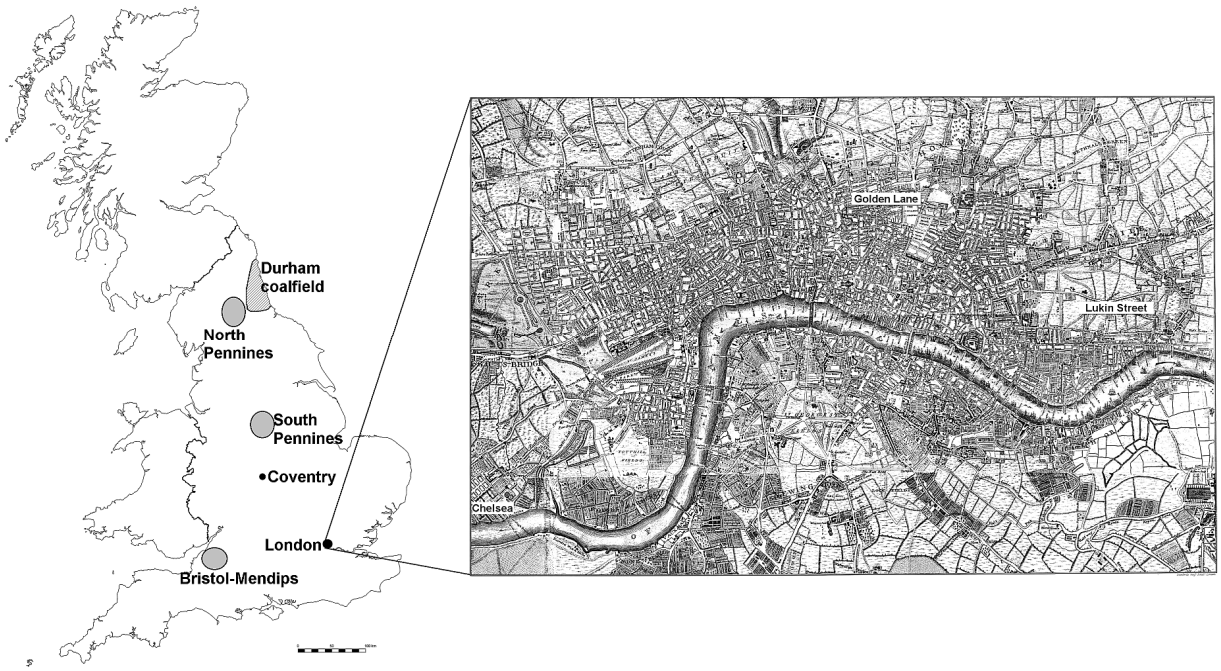

Figure 2

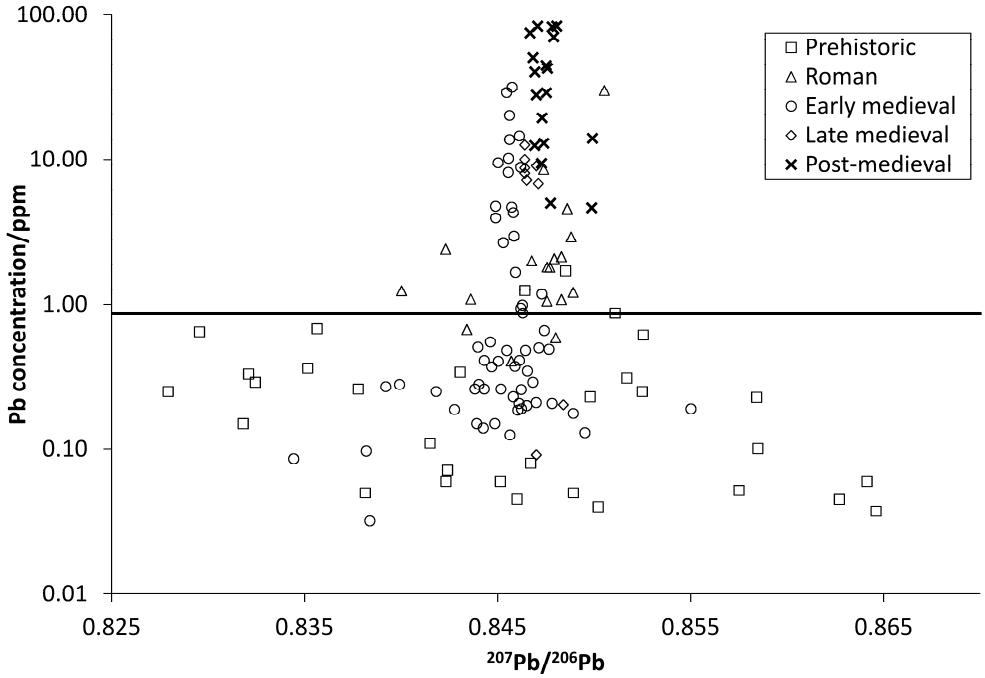

Figure 3

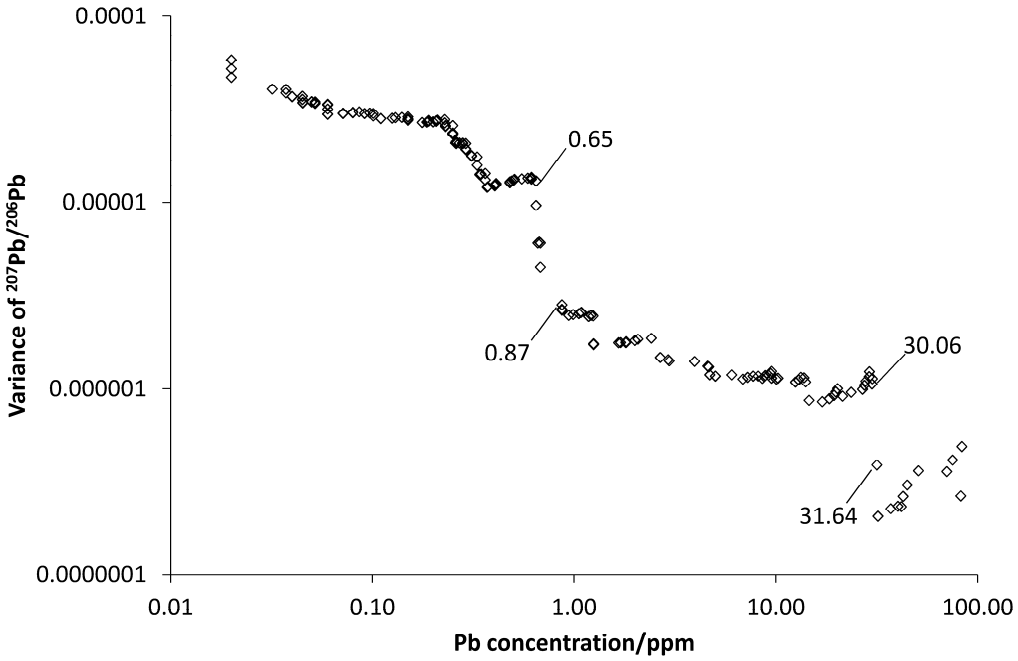

Figure 4